Canadian Soil Quality Guidelines for Contaminated Sites

Human Health Effects:

INORGANIC ARSENIC

Final Report

The National Contaminated Sites Remediation Program

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NOTICE

This document provides human health soil quality guidelines for contaminated sites in Canada. It is issued in support of the National Contaminated Sites Remediation Program. The values in this document are general guidance only. They do not establish a binding norm, or prohibit alternatives not included in the document. They are not finally determinative of the issues addressed. Decisions in any particular case will be made by applying the law and regulations of the basis of specific facts when regulations are promulgated or permits are issued.

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1. INTRODUCTION

The Canadian Council of Ministers of the Environment's (CCME) Canadian Soil Quality Guidelines for Contaminated Sites are numerical limits for contaminants in soil and water intended to maintain, improve, or protect environmental quality and human health at contaminated sites in general. In response to the urgent need to begin remediation of high priority "orphan" contaminated sites, an interim set of criteria was adopted from values currently in use in various jurisdictions across Canada (CCME 1991).

Many of the CCME (1991) interim soil remediation criteria do not have a complete supporting rationale. This document provides the supporting scientific rationale required to accept or modify the interim soil criteria as final environmental quality criteria for arsenic.

This report reviews the sources and emissions of arsenic, its distribution and behaviour in the environment and its toxicological effects on humans and experimental animals. Human health soil quality guidelines are defined for residential/parkland, agricultural (limited to residential uses of farm property), commercial and industrial land uses and the use of ground water as a source of water for drinking. In addition, various check mechanisms considering indirect pathways of exposure (agricultural produce, milk and meat contamination, backyard produce contamination, and offsite migration of contaminants via wind and water erosion) are elaborated in the *Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines* (CCME 1996); these check mechanisms may be used to adjust the human health soil quality guidelines derived herein, if desired.

The values derived herein are human health soil quality guidelines and are intended as general guidance. Site specific conditions should be considered in the application of these values. The values may be applied differently in various jurisdictions, therefore the reader should consult the appropriate jurisdiction for application of the values.

The supporting documentation and assessment contained in this report were extracted and adapted from HWC (1992), EC/HWC (1993), Health Canada (1993), and Hughes *et al.* (1994).

2. BACKGROUND INFORMATION

2.1 Physical and Chemical Properties

Arsenic is a metalloid of the group VA elements (N, P, As, Sb, Bi) which exhibits both metallic and non-metallic properties. The atomic number and atomic weight of arsenic is 33 and 74.92, respectively. Arsenic possesses three unpaired electrons in its outer shell which allows it to have unusual electrical and spectroscopic properties (NRCC 1978). Chemically, arsenic closely resembles phosphorus (NRCC 1978). Arsenic can occur in four oxidation states as arsine (-3), arsenic metal (0), arsenite (+3), and arsenate (+5). Arsenic bonds covalently with most non-metals and metals. It forms stable organic compounds in both its trivalent and pentavalent states. Some physical and chemical properties of arsenic and inorganic arsenic compounds are presented in Table 1.

Arsenic, in various forms occurs naturally in most environmental compartments. It is rarely found in its elemental form (Slooff *et al.* 1990). Naturally occurring arsenic is present most commonly as sulphides and as complexes with iron, nickel, copper and cobalt (Woolson 1975). The anthropogenic form of arsenic most frequently released to the environment is arsenic(III) oxide (Slooff *et al.* 1990).

2.2 Analytical Methods

A review of the analytical chemistry of arsenic is beyond the scope of this document but can be found in publications by Talmi and Feldman (1975), Irgolic *et al.* (1983), Korte and Fernando (1991) and most recently CCME (1993a,b).

In soils, the fraction of the total arsenic which is of relevance is the 'available' arsenic and the amount that is rendered available is dependent upon environmental conditions. Therefore, soils of differing physical and chemical characteristics with the same soil arsenic concentrations will exhibit varying levels of plant-available arsenic and phytotoxicity. Extraction of arsenic from soils has followed the use of separate media extraction or the fractionation approach common to soil phosphorus analysis. No one extractant will serve as a primary index of all plant available arsenic. The extractant selected should be specific for certain arsenic components in the soil.

One method of analysis for total arsenic in soil has been recommended by the CCME (1993a,b) in an effort to promote consistency within the National Containated Sites Remediation Program (NCSRP). U.S. EPA Method 6010 (Inductively Coupled Plasma-Atomic Emission Spectroscopy) prescribes a sequential sample preparation using nitric acid, hydrogen peroxide and hydrochloric acid and followed by analysis by an inductively coupled argon plasma emission spectrometer capable of background correction. The estimated instrumental detection limit for the method is 53 µg/L (or 5.3 µg/g following corrections for 1 g soil sample digestion with the extract being diluted to 100 mL).

2.3 Production and Uses in Canada

Arsenic occurs as a minor constituent in complex ores which are mined primarily for their copper, lead, zinc, silver and gold content. In Canada, gold ores are the main source of arsenic, whereas worldwide, copper ore is the predominant source. Arsenic is usually recovered from dusts and residues associated with the roasting of these ores and is collected as impure arsenic trioxide, and is either purified on site or sold directly to a refiner. Ninety-six percent of arsenic is utilized as arsenic trioxide or, other arsenic compounds and only 4% is utilized in the metallic state. It is produced mainly as arsenic trioxide through the roasting of arsenious gold ores. Though four gold-ore roasters operated in the 1970s, only two mines produced arsenic in 1992. Demand for arsenic has fallen sharply since the early 1980s, due to concerns about environmental effects. Because of low prices, arsenic trioxide currently produced in Canada is being stockpiled for future sale (Ignatow *et al.* 1991).

At present in Canada, arsenic is used mainly in metallurgical applications (Environment Canada 1991) and in the manufacture of wood preservatives. For the period 1984 to 1988, Canadian sales of arsenic (as arsenic pentoxide) for use in wood preservation fluctuated between about 650 and 1300 tonnes (t) per year (E. Brien, Environment Canada, unpublished data). Arsenic compounds have also been used in Canada in herbicides (E. Brien, Environment Canada, unpublished data), in pharmaceuticals and in glass manufacturing (CCREM 1987). World production of arsenic trioxide was 56,000 t in 1990. This was an increase of approximately 19 000 t since the early 1980s (Law-West 1987; Ignatow *et al.* 1991). In contrast, the demand for arsenic in Canada has decreased significantly since the early 1980s because of environmental concerns about its use in agriculture. Canadian production of arsenic trioxide in 1985 was approximately 8 400 t whereas in 1986 it had decreased to 5 100 t (Law-West 1987).

In 1990, approximately 70% of the world's arsenic was used in wood preservatives, 22% in agricultural chemicals, 4% in glass manufacturing, 2% in nonferrous alloys and 2% in other uses. Over 50% of the world's arsenic production is consumed by the United States (Ignatow *et al.* 1991).

Wood Treatment

Arsenical wood preservatives are used in order to prevent rotting or insect damage in building foundations, fence posts, submerged footings and utility poles. The most common arsenic compounds used are chromated copper arsenate (CCA), ammoniacal copper arsenate (ACA) and fluor chrome arsenate phenol.

Agriculture

Arsenical agricultural chemicals are used as herbicides, plant desiccants and defoliants to control grassy and broadleaf weeds. Since the 1970's, the inorganic arsenical herbicides such as lead arsenate and calcium arsenate have been replaced with the less phytotoxic organic arsenical herbicides. Monosodium

methylarsonate, disodium methylarsonate and dimethylarsinic acid are the most commonly used agricultural arsenical compounds (Wauchope and McDowell 1984; Adriano 1986; Ignatow *et al.* 1991).

Glass Manufacturing

Arsenic trioxide is used in the glass industry as a decolorizing agent. It is also used to remove tiny air bubbles in the glass (Ignatow *et al.* 1991).

Nonferrous Alloys and Other Uses

Arsenic metal is used as a minor alloying agent in certain copper- and lead-based alloys. Arsenic increases the corrosion resistance and tensile strength of copper used in industrial plant piping and auto radiators. Gallium arsenide and its alloys are employed as semi-conductors which are then used in such products as light emitting diodes, microwave devices, solar cells and photo-emissive surfaces (Ignatow *et al.* 1991).

In 1974, the most recent account of As use in Canada, 240 t of arsenic were used: 67% in glass making; 32% in metal rolling, casting and extruding; and the rest in other chemical industries (CCREM 1987). There is no doubt that patterns of use have changed since these figures were reported. Konasewich and Henning (1988) for example, have stated that in 1984, 5 000 t of 50% CCA concentrate (about 550 t of arsenic) were used in Canada for wood preservation.

2.4 Environmental Fate and Behaviour (Extracted from Environment Canada 1995)

As Woolson (1983) has noted, the ultimate sink for most environmental arsenic is ocean sediment. Because of its reactivity and mobility, however, arsenic can cycle extensively through both biotic and abiotic components of local aquatic and terrestrial systems, where it can undergo a variety of chemical and biochemical transformations, such as oxidation, reduction, methylation and demethylation (Cullen and Reimer 1989).

The following sections describe the major fate and behaviour processes of arsenic in the environment.

Soil

Many factors control the fate and behaviour of arsenic in soil, and ultimately its bioavailability. It is generally accepted that only soluble arsenic is available for plant uptake. The solubility and speciation of arsenic in solution are primarily determined by pH and the reduction/oxidation potential (Eh). The solubility of arsenic may determine the amount of arsenic ultimately available within a system while the speciation of arsenic determines the behaviour and to a large extent the toxicity of arsenic.

Transformations

Generally, in oxidizing conditions or aerobic soils, inorganic arsenic is predominantly present as arsenate, AsO_4^{-3} with arsenic in the +5 oxidation state. In this environment arsenic acid can dissociate into four different species depending upon the pH (Eq. 1).

$$H_3AsO_4 \leftrightarrow H_2AsO_4^- \leftrightarrow HAsO_4^{2-} \leftrightarrow AsO_4^{3-} \qquad \text{Eq. 1}$$

Thermodynamically, $H_2AsO_4^-$ is the most stable species between pH 2 and pH 7 which overlaps with the normal soil pH range of 4 to 8. Above pH 7 $HAsO_4^{2-}$ is the most stable.

Under reducing conditions, as in anaerobic, water-logged soils, arsenic is primarily present as arsenite, AsO_3^{3-}, with arsenic in the +3 oxidation state (O'Neill 1990). Arsenous acid dissociates into four pH dependant species in the same manner as arsenic acid (Eq. 2).

$$H_3AsO_3 \leftrightarrow H_2AsO_3^- \leftrightarrow HAsO_3^{2-} \leftrightarrow AsO_3^{3-} \qquad \text{Eq. 2}$$

H_3AsO_3 is thermodynamically the most stable species up to about pH 9 and is the predominate species within the normal soil pH range. Under strongly reduced conditions elemental arsenic and arsine (AsH_3) can exist (Elkhatib *et al.* 1984a,b).

Alterations in the oxidation state of arsenic can greatly affect its solubility in soil. At high soil redox levels (500-200mV), arsenic solubility is low and most (65 -98%) of the arsenic in solution is present as As(V). In alkaline, or reducing conditions, a substantial proportion of arsenic can be released into solution. Also, under moderately reducing conditions (0-100 m), arsenic solubility can be controlled by the dissolution of iron oxyhydroxides (Masscheleyn *et al.* 1991).

Soil pH and Eh can act together to influence arsenic speciation. For example, in sediment, under anaerobic conditions at pH 5.0 and 6.5, all soluble arsenic residues are in the trivalent state, while at pH 8.0, at the same redox conditions, 13.9% of the soluble arsenic is found to be in the pentavalent state (Brannon and Patrick 1987). Furthermore, in alkaline soils, a lower redox potential is required for the reduction of arsenate to arsenite than in acid soils (Slooff *et al.* 1990).

A variety of microorganisms can methylate arsenic (Masscheleyn *et al.* 1991). Methylated arsenic oxyacids are found in various natural waters, soils and sediments. Some of these methylated arsenic compounds include monomethylarsonic acid, $CH_3AsO(OH)_2$, dimethylarsinic acid, $(CH_3)_2AsO(OH)$, and trimethylarsenic oxide, $(CH_3)_3AsO$. Methylation to produce the volatile compounds, trimethylarsine, $(CH_3)_3As$, and dimethylarsine, $(CH_3)_2AsH$, has been observed (Masscheleyn *et al.* 1991).

Biomethylation depends upon both the microorganisms and the arsenic compounds present. Some microorganisms can fully methylate arsenic compounds over a wide range of pH conditions, whereas other microorganisms are more limited in the degree to which they can perform methylation (O'Neill 1990). Conditions which promote microbial activity such as high organic matter content, warm temperatures and adequate moisture often enhances methylation of arsenicals (Woolson 1977).

Fungi, moulds and various strains of bacteria can also demethylate arsenicals. Demethylation of organic arsenicals by these microorganisms occurs through oxidative pathways in which methyl groups are cleaved and oxidized to CO_2 resulting in the production of inorganic arsenate (Bibhas 1975).

Adsorption

One of the most important processes influencing the behaviour and bioavailability of arsenic in soil is its ability to sorb onto solids or particulates. Soil sorption controls the persistence, activity and movement of arsenic species (Wauchope and McDowell 1984). Factors governing the sorption of arsenic in soil include pH and the amount of clay, iron, aluminum, calcium, and phosphorus present (Woolson *et al.* 1971).

The pH influences the distribution of arsenic species present in solution and activates the anion adsorption sites on clay minerals thus affecting adsorption. At low pH, hydroxyl groups exposed on the periphery of metal oxides, clay minerals and amorphous silicate minerals are protonated and acquire a positive charge. These protonated (activated) sites are then available for interaction and adsorption of the arsenical anions present in solution (Sposito 1989).

Arsenic is strongly adsorbed by clay minerals such as kaolinite, limonite, montmorillonite and vermiculite (Dickens and Hiltbold 1967; Slooff *et al.* 1990). Adsorption capacities for arsenate in a variety of soils were rated as follows: clay soils > calciferous soils > peat soils > acid woodland > podzol soils > sandy soils (Slooff *et al.* 1990). Arsenic can be strongly sorbed to electropositive hydroxides such as aluminum, iron and calcium which coat clay particles or react with cations in the soil solution (Jacobs *et al.* 1970).

Some studies have reported that the insoluble complexes between iron(III) hydroxides and arsenic strongly influence arsenic soil adsorption, while other studies such as Johnson and Hiltbold (1969) found much of

the particulate bound arsenic in soils complexed with aluminum. There is evidence, however, that aluminum hydroxides act as the most active adsorbent initially, but iron hydroxides are more favoured over time as equilibrium is reached (Woolson 1973). Livesey and Huang (1981) reported that poorly ordered Al and Fe compounds, and particulary Al components, are closely associated with the arsenic adsorption by soils.

Persistence/Movement

The extensive use of arsenic pesticides in early pest control programs has increased the residual content in certain soils. For example, arsenic residues of 440 µg/g have been reported in orchard soils with a history of lead arsenate application. One study has suggested that the entire application of lead arsenate ($PbHAsO_4$) to an orchard soil was still in the top 25 cm of the soil 15 years later (Aten *et al.* 1980).

Arsenic residues in browse and herbaceous vegetation in douglas fir, ponderosa pine and western larch forests in which trees were treated with monosodium methyl arsonate (MSMA) for thinning were relatively low. Large quantities of arsenic did not move from the forest floor into the soil, indicating that arsenic residues were tightly bound in the foliage. Only small amounts of arsenic were detected in streams near forests treated with MSMA or cacodylic acid (CA or dimethylarsinic acid) (Norris *et al.* 1983).

Inorganic arsenicals used as soil sterilants, such as sodium arsenite, are persistent in the soil and may remain effective for periods as long as five to eight years especially in areas of low rainfall (White-Stevens 1971).

Leaching

Inorganic arsenic is more mobile than organic arsenic in soil and arsenite is more mobile than arsenate (Xu *et al.* 1988). Thus, inorganic arsenic and arsenite might leach into surface and ground waters (NRCC 1978). Despite the relative stability of soluble ionic species, concentrations of arsenicals in porewaters are usually low (O'Neill 1990). It seems that adsorption dominates the partitioning of arsenic to the particulate phase, hence leaching of arsenicals at greater soil depths is well correlated with the decreasing adsorptive capacities of the soil. Essentially, leaching occurs when arsenical application rates exceed the adsorption capacity of the soil. Leaching is pronounced in sandy soils and soils lacking in hydroxides of iron, aluminum and calcium (Woolson *et al.* 1971).

Bioaccumulation

Though plants grown in arsenic-treated soil may contain considerably greater concentrations of arsenic than plants grown in untreated soils, terrestrial plants rarely contain higher concentrations of arsenic than their substrate (Walsh and Keeney 1975). Grasses growing on contaminated spoil tips from old mines in south western England were reported to contain concentrations as high as 3 460 mg As/kg of arsenic but still did not reach the 26530 mg As/kg concentrations present in the soil (Porter and Peterson 1977)

In contrast, the extended metabolic abilities of aquatic macrophytes allows considerable accumulation of arsenic from the water. Lunde (1973) reported arsenic bioconcentration factors of 250 to 3000. Woolson (1975) reported that many species of marine plants and animals contain naturally high concentrations of arsenic, which is often in the organic form and substantially less toxic than other forms. Metabolism in aquatic plants has been described by Lunde (1973), Cooney *et al.* (1978) and Nissen and Benson (1982).

Interactions with Other Elements

Soil solutions contain several different constituents that are capable of competing for adsorption sites (Roy *et al.* 1986), particularly phosphorus, selenium and molybdate. Arsenic and phosphorus are chemically similar and exist as oxyanions which may compete for fixation sites in soil and for uptake in plants (Woolson 1973; Asher and Reay 1979).

Studies on interactions of arsenic with selenium and molybdenum have shown antagonistic action on arsenic uptake by plants. Arsenic, selenium and molybdenum can compete as inorganic and organic ligands in the soil solution phases (Khattak *et al.* 1989).

2.5 Levels in the Canadian Environment

Base-metal and gold-production facilities, the use of arsenical pesticides, coal-fired power generation and the disposal of domestic and industrial wastes, are the principal anthropogenic sources of arsenic released into the Canadian environment. Most (80%) of the anthropogenic releases to the environment are ultimately released to soil (ATSDR 1993). Arsenic is present naturally in the aquatic and terrestrial environments from weathering and erosion of rock and soil. In areas of arsenic-enriched bedrock, background concentrations can be significantly elevated. Arsenic is released naturally into the atmosphere by volcanic eruptions and the escape of volatile methylarsines from soil (Chilvers and Peterson 1987).

The following section contains an overview of concentrations of arsenic in soil, air, water, and sediment in Canada.

Soil

Worldwide, there is a range in natural arsenic content in uncontaminated soils. Walsh and Keeney (1975) reported arsenic concentrations in uncontaminated soils between 0.2 and 40 µg/g. However, Kabata-Pendias and Pendias (1984) have reported a range of <1 to 95 µg/g with a mean arsenic concentration for world soils of 8.7 µg/g (Kabata-Pendias and Pendias 1984; Adriano 1986). The average arsenic concentration, worldwide, in both soil and rocks, has been estimated at 3 µg/kg (Boyle and Jonasson 1973). It has been noted that arsenic concentrations may vary greatly depending on the type of soil and the hydrology (Slooff *et al.* 1990). Arsenic is found primarily in its pentavalent form in soil (Amdur *et al.* 1991).

Data from recent surveys undertaken by the Geological Survey of Canada (see Table 2a) demonstrate that the "natural background" concentrations of arsenic in surficial media such as glacial tills (the substrate on which most Canadian soils are developed) span over several orders of magnitude with concentrations ranging from 1 to 2,704 µg/g dry wt (range of medians: 2 to 54 µg/g dry wt). The wide range of concentrations reflects changes in the bedrock geology and the effects of glacial erosion of bedrock debris (I. Kettles, NRCan, pers. com.). The concentrations of arsenic in surface soil found at various uncontaminated sites across Canada are summarized in Table 2b. Kabata-Pendias and Pendias (1992), in a review of available data, report that mean concentrations of arsenic in several uncontaminated soil types in Canada range from 4.8 to 13.6 µg/g dry weight (dw). On examination of adequate data sets found in Table 2b, average urban and agricultural concentrations were found to be at or below 10 µg As/g dry soil with most of the values in the 4 to 6 µg/g range. Background arsenic levels have been provisionally established for Ontario soils at 11 µg/g for agricultural lands and 17 µg/g for residential and parkland areas (OMEE 1994b). Background levels, as calculated by OMEE (1994b), consist of the value of the 98th percentile plus twice the value of the coefficient of variation (L. Pastorek, OMEE, pers. com.).

Slightly elevated levels of arsenic have been reported in acid sulphate soils where arsenic is associated with pyrite. Dudas (1987) measured concentrations of 23 to 40 mg/kg in two acid sulphate soils in Alberta. Much higher concentrations (see Table 2c) were found near base-metal smelters (mean concentrations ranged from 50 to 110 mg As/kg, with a maximum of 2 000 mg As/kg) (Temple *et al.* 1977; Hertzman *et al.* 1991; Brunswick Mining and Smelting Corporation, pers. com.; Murphy, unpublished data; Teindl, unpublished data) and gold-mining/roasting operations (mean concentrations of 60 to 110 mg As/kg, and maximum of >10 000 mg As/kg) (Gemmill 1977; NRCC 1978; Hutchinson *et al.* 1982; EBA Engineering Consultants Ltd. 1991). Large amounts of arsenic have also been reported in soil, sediment and water in the vicinity of arsenic-bearing precious metal deposits near Cobalt, Ontario, and Halifax, Nova Scotia (Boyle *et al.* 1969; Brooks *et al.* 1982; Bottomley 1984). Elevated arsenic levels have also been reported in soils where arsenical pesticides (including wood-preservation compounds) have been used; for example, concentrations

of arsenic of up to 290 mg/kg (mean values of up to 54 mg/kg) have been detected in soil from orchards in Ontario (Boyle and Jonasson 1973; Frank *et al.* 1976), and up to 10 860 mg/kg (mean values of up to 6 000 mg/kg) at active wood-preservation facilities in Atlantic Canada (Bamwoya *et al.* 1991). Highest arsenic concentrations (up to 75 000 mg/kg; typically 3 000 to 4 000 mg/kg) were found in tailings at base-and precious-metal mine sites in Ontario and Nova Scotia (Hawley 1980; Dale and Freedman 1982). Table 2c summarizes the concentrations of arsenic in contaminated surface soils from various sites across Canada. Analysis of whole-soil and soil pore water from a limited number of areas indicates that most (>90%) of the arsenic in soils is inorganic (Woolson 1983; Haswell *et al.* 1985; Masscheleyn *et al.* 1991).

Ground Water

Ground water normally contains higher concentrations of arsenic than are found in associated surface water (Boyle and Jonasson 1973). Available data indicate that most Canadian ground waters contain less than 50 µg As/L (Michel 1990; Léger 1991); however, concentrations of arsenic have been reported to range up to 9 100 µg/L in ground water in areas with a high content of arsenic in the bedrock, such as in regions of Ontario, Quebec, New Brunswick and Nova Scotia (Boyle *et al.* 1969; Grantham and Jones 1977; Lalonde *et al.* 1980; Bottomley 1984; Méranger *et al.* 1984; Deveau, pers. com.). Gold mining activities in Nova Scotia have been reported to contribute to high arsenic levels in local ground waters (Bottomley 1984). High arsenic concentrations (up to 11 000 µg/L) were also detected in ground water in the vicinity of an abandoned arsenical wood preservative facility near Vancouver, British Columbia (Henning and Konasewich 1984). Recent studies (Masscheleyn *et al.* 1991; Korte 1991) in other areas suggest that arsenic in ground water is present mainly as inorganic As(III) and As(V) species.

Sediment

In Canada, background concentrations in relatively uncontaminated surface sediment are generally less than 20 µg As/g dry weight; arsenic levels in deeper sediment are normally only a few µg/g (Friske 1985; Johnson 1987). Arsenic accumulation (up to 65 µg/g) has been reported in contaminated Halifax harbour sediments (Tay *et al.* 1991). Bamwoya *et al.* (1991) found 262 µg As/g in sediment downstream from an arsenical wood preservation facility near Elmsdale, Nova Scotia. Concentrations of arsenic are highest in sediments near base- and precious-metal mining and ore-processing operations. Average levels of 100 to 200 µg/g (maximum 650 µg/g) were reported near base- metal mines and smelters in several provinces (Franzin 1984; Bailey 1988; Palmer *et al.* 1989). Near gold mines and an abandoned precious-metal refinery, mean concentrations in sediments ranged from about 700 to 5 000 µg As/g (maximum 18 650 µg/g) (Trip and Skilton 1985; Sutherland 1989; Diamond 1990; Reimer and Bright, unpublished data). Limited data on the composition of pore waters from two areas in Canada (Reimer and Thompson 1988; Diamond 1990) suggest that most (≥85% of) bioavailable arsenic in sediment is present as inorganic As(III) and As(V).

Surface Water

Arsenic levels in Canadian surface waters remote from point sources of contamination are typically < 2 µg/L (Bailey 1988; Environment Canada 1988; Belzile and Tessier 1990; Traversy *et al.* 1975). Elevated concentrations of arsenic have been reported in surface waters in the vicinity of gold-mining or ore-roasting operations. Mean levels of about 45 µg/L (maxima about 140 µg/L) were found near abandoned gold mines in Nova Scotia (Mitchell Brook) and Ontario (Moira Lake) during the 1980s (Brooks *et al.* 1982; Diamond 1990). Highest arsenic levels were found in samples of water collected from several lakes near the gold mines and roasters at Yellowknife in the mid-1970s; for example, Keg and Kam Lakes contained from 700 to 1 500 µg As/L and 1 500 to 5 500 µg As/L, respectively
(CPHA 1977; Wagemann *et al.* 1978). Limited data from the early 1990s indicated that concentrations in these lakes had declined appreciably, to about 545 and 645 µg/L in Keg Lake and Kam Lake, respectively (Reimer and Bright, unpublished data). Most (≥80%) of the arsenic in contaminated waters, as well as in surface waters remote from point sources, is expected to be present as inorganic As(V) and to a lesser extent As(III) species (Cullen and Reimer 1989).

Drinking Water

Data on concentrations of arsenic in Canadian drinking water from a nationwide survey have not been identified. Based on data obtained in provincial monitoring programs, levels in Canadian drinking water from both surface- and ground water supplies are generally less than 5 µg/L (measured as total arsenic), although concentrations in some ground water supplies are greater (Environment Canada 1989a; 1989b; 1989c; 1989d; OME 1989; Manitoba Environment 1989). Five percent of samples of well water from contaminated supplies in Nova Scotia contained more than 500 µg As/L (Méranger *et al.* 1984).

Ambient Air

The two principal natural sources of atmospheric arsenic are biological methylation and volatilization following weathering processes. Worldwide, natural atmospheric emissions of arsenic are approximately 50% greater than anthropogenic emissions (NRCC 1978; Slooff *et al.* 1990). The mean atmospheric arsenic concentration is 1 ng/m^3 over land and ranges from 0.02 to 0.16 ng/m^3 over oceans (Walsh *et al.* 1979). Most of the arsenic in air is associated with fine suspended particulates of less than 5 µm in diameter.

In Canada, recent long-term studies undertaken by Environment Canada indicate low arsenic concentrations (in PM_{10}, inhalable particulates of <10 µm in diameter) in ambient air in 15 urban areas and 4 rural sites. Between 1986 and 1993, 53% of 3,863 ambient air samples from across Canada contained arsenic in concentrations below the detection limit of 0.0005 µg/m^3; a mean concentration of 0.001 µg/m^3 was calculated incorporating all data points and substituting half the detection limit for values below detection (Dann 1994). NRCC (1978) reported that total arsenic concentrations ranged between 0.27 and 4.7 ng/m^3 in Canadian rural areas.

Arsenic concentrations in ambient air near mines and smelters vary depending on the sampling distance from these sites. Between 1973 and 1975, the concentration of arsenic in suspended particulates, near Yellowknife gold smelters, ranged from < 10 to 3910 ng/m^3. Mean concentrations were between 60 and 90 ng/m^3 with arsenic concentrations near Giant Yellowknife mine (NWT), Con Mine (NWT) and Yellowknife (NWT) of 1680 ng/m^3, 380 ng/m^3, and 391 ng/m^3, respectively (Gemmill 1977).Atmospheric arsenic near such industrial sources is typically As_2O_3 in particulate form (Cullen and Reimer 1989).

Indoor Air and House Dust

There is a paucity of information on levels of arsenic in indoor environments such as air and house dust. However, based on limited data and information, the levels of arsenic measured indoors appear to be comparable to those measured outdoors.

Bell *et al.* (1994) screened indoor air samples from 37 homes of the Windsor area for the presence of arsenic and other contaminants using Lo-Vol sampling methodology (low volume, 25 L/min). Filters were digested in weak (5%) nitric acid and analyzed using ICP-MS (Inductively Coupled Plasma-Mass Spectrometry). The levels of airborne arsenic ranged from 0.3 to 4.3 µg/m^3 with a mean of 0.7 µg/m^3 and a median of 0.5 µg/m^3. Slightly higher levels of arsenic were measured concurrently outdoors (residential and commercial areas of Windsor) with a mean of 1.3 µg/m^3 (N= 46 samples, range: 0.4 - 3.3).

In a recent survey performed for the Bureau of Chemical Hazards of Health Canada, house dust and yard soil samples from 50 Ottawa homes were analyzed for arsenic and other trace elements by ICP-MS after digestion with HF/HNO_3 (Kean 1995). Preliminary results indicate that house dust and yard soil to contain geometric mean concentrations of 4.76 µg As/g dust (range: 1.70-79.47) and 2.88 µg As/g soil (range: 1.70-9.90), respectively.

Commercial Food

Arsenic has been detected in most foodstuffs consumed by humans; however, the proportion of inorganic arsenic varies considerably. Much of the arsenic in fish, for example, is present in highly complexed forms that are not bioavailable, or as organic compounds that are rapidly excreted from the body. The percentage of total arsenic that is inorganic in various foods has been determined to range from 0% in saltwater fish to

75% in milk, dairy products, beef and pork, based on limited data (Weiler 1987). Levels of total arsenic were recently determined in 112 food composites from 6 Canadian cities between 1985 and 1988 under the Total Diet Program (Dabeka *et al.* 1993). The highest mean concentrations of total arsenic in 7 collections were found in fish (1662 ng/g), meat and poultry (24.3 ng/g), bakery goods and cereals (24.5 ng/g), and fats and oils (19.0 ng/g). Lower mean concentrations of total arsenic were determined in an earlier survey of 10 food groups in a duplicate diet study in five Canadian cities (range: 0.46 ng/g in drinking water to 60.1 ng/g in meat, fish and poultry (Dabeka *et al.* 1987). Based on very limited data, concentrations of arsenic in produce grown in the vicinity of industrial sources may be somewhat higher than those reported in the duplicate diet study (Teindl, unpublished data; Noranda Mines 1992; Brunswick Mining and Smelting Corporation, pers. com.; Murphy unpublished data).

Biota Used as Human Food

Levels of arsenic in the tissue of freshwater fish in areas remote from point sources of contamination generally range from < 0.1 to 0.4 μg As/g fresh weight (fw) (Moore and Ramamoorthy 1984). Concentrations in samples of fish in Lakes Erie and Ontario in the mid 1970s and in base-metal mining areas in Atlantic Canada in 1984 and 1985 were usually within this range (Traversy *et al.* 1975; Bailey 1988). Higher levels were reported in fish sampled near active and abandoned gold-mining operations, with maximum concentrations ranging from 2.36 to 4.77 μg As/g (fw) (Gemmill 1977; Azcue 1992; Dale and Freedman 1982).

Human Tissues and Body Fluids

Mean levels of arsenic in human breast milk in India range from 0.3 to 0.6 ng/g (Dang *et al.* 1982). Arsenic has been detected in human breast milk in Yugoslavia, with a mean concentration of total arsenic of 1.3 ng/g (range: 0.2 - 3.8 ng/g) (Kosta *et al.* 1983).

Urinary arsenic concentrations in "unexposed" populations are normally below 50 µg/L, based on a review of values considered "normal" in earlier reports (Landrigan 1981). Although in some earlier studies, control for external contamination of hair samples by arsenic was inadequate, concentrations in urine and hair are often greater in individuals living in the vicinity of point sources, such as smelters, than in comparison populations (Milham and Strong 1974; Polissar *et al.* 1990; Baker *et al.* 1977), and in persons whose diets frequently contain fish and seafood (Vahter 1986). Conversely, however, Jensen *et al.* (1991) reported that there was no significant difference in the concentration of arsenic in the urine of children and adults residing in a "chemically polluted area" (former sites of industries which used arsenic) of Denmark, compared to that of those living in an unpolluted area. Smokers are reported to have 50% higher mean arsenic levels in blood than in non-smokers (Kagey *et al.* 1977). Median arsenic levels in various other tissues in the general population and in "exposed" individuals ranged from 0.001 µg/g in the heart to 3.0 µg/g in nails, based on a summary of several reports (Fergusson 1990).

2.6 Existing Criteria and Guidelines for Arsenic in Various Media

Existing criteria, guidelines or standards for arsenic in soil and water from provincial and international agencies are summarized in Tables 3.

3. BEHAVIOUR AND EFFECTS IN HUMANS AND EXPERIMENTAL ANIMALS

3.1 Human Exposure Estimates (Adapted from Hughes *et al.* 1994)

Estimated daily intakes (on a per kg body weight basis) of inorganic arsenic from environmental media by various age groups in the Canadian population are presented in Table 4. Ingestion in water and food represent the principal routes of inorganic arsenic intake for all age groups, followed by soil/dust in infants and children, and water and air for all age groups. Based on limited data on the relative proportion of

inorganic arsenic in various foodstuffs, average daily intake from food ranges from <0.08 to 0.3 µg/kg bw/day for adults and young children, respectively. Intake in soil is estimated to range from 0.003 µg/kg bw/day in adults to 0.06 µg/kg bw/day in young children. Based on the limited monitoring data on the concentration of arsenic in the drinking water of Canadians, average daily intake of arsenic, which in surface-water supplies is predominantly in the pentavalent inorganic form, in this medium by each age group is usually less than 0.5 µg/kg bw/day; however, intake of inorganic arsenic (both As(III) and As(V)) from drinking water may be higher from some ground water supplies. Average daily intake of arsenic from ambient air is estimated to range from 0.0003 to 0.0004 µg/kg bw/day. Total daily exposure to inorganic arsenic from environmental sources ranges from 0.18 to 0.73 µg/kg bw/day, with the greatest exposure being in infants (Table 4). Cigarette smoking may contribute an additional 0.01 to 0.04 µg/kg bw/day.

3.2 **Pharmacokinetics** (Extracted from HWC 1992)

Ingested elemental arsenic is poorly absorbed and largely eliminated unchanged (HWC 1989). Arsenic oxides are readily absorbed (>80%) from the gastrointestinal tract (Fowler *et al.* 1979) and, to a lesser extent, through the lungs and skin (Wickstroem 1972). On the basis of faecal recovery experiments in human volunteers, the absorption of soluble As(III) and As(V) compounds is close to 95% (Hindmarsh and McCurdy 1986). Arsenic(III) tends to accumulate in the tissues, whereas As(V) and organic arsenic are well absorbed but rapidly and almost completely eliminated via the kidneys (Bertolero *et al.* 1987).

Following ingestion, inorganic arsenic appears rapidly in the circulation, where it binds primarily to haemoglobin (Axelson 1980); within 24 hrs, it is found mainly in the liver, kidneys, lungs, spleen and skin (Wickstroem 1972). Skin, bone and muscle represent the major storage organs. The accumulation of arsenic in skin is probably related to the abundance of proteins containing sulfhydryl groups, with which arsenic readily reacts (Fowler *et al.* 1979). In humans, inorganic arsenic does not appear to cross the blood-brain barrier; however, transplacental transfer of arsenic in both humans (Gibson and Gage 1982) and mice (Hood *et al.* 1987) has been reported.

There appear to be two main processes, with different rates, for the elimination of ingested trivalent arsenic from the body (Lovell and Farmer 1985). The first is rapid urinary excretion of non-methylated arsenic in both the trivalent and pentavalent forms (close to 90% of the total urinary arsenic over the first 12-hour period). The second involves detoxification by sequential methylation of As(III) in the liver to dimethylarsinic acid (DMAA) (Lovell and Farmer 1985; Butchet and Lauwerys 1985). Excretion of the methylated compounds commences approximately five hours after ingestion but reaches its maximum level two to three days later. The half-life of inorganic arsenic in humans is estimated to be between two and 40 days (Pomroy *et al.* 1980).

The results of a study in which inorganic arsenic (125, 250, 500 or 1,000 µg $NaAsO_2$) was administered orally once a day for five consecutive days to four volunteers indicate that the arsenic methylation capacity is progressively saturated when daily intake exceeds 0.5 mg (Buchet *et al.* 1981a); it does not, however, appear to be completely saturated even for daily doses as high as 1 mg. Studies with human volunteers indicate that most ingested organic arsenic is rapidly excreted unchanged (>80% of the dose within 4 days) (Buchet *et al.* 1981b; Luten *et al.* 1982; Tam *et al.* 1982).

3.3 **Essentiality** (Extracted from EC/HWC 1993)

It has been postulated that arsenic might be an essential element in animals, as there evidence that such essentiality is plausible, but there is no indication that arsenic is essential to humans (U.S. EPA 1988).

3.4 **Toxic Effects** (Unless otherwise specified, this section was extracted from EC/HWC 1993)

Acute and Short Term Toxicity

The acute toxicity of inorganic arsenic compounds increases with solubility in water. Soluble salts, such as sodium and potassium arsenates and arsenites, arsenic pentoxide, arsenic trichloride and arsenic acid, are highly acutely toxic to mammals, with LD_{50}s in rats, mice and rabbits ranging from 2.6 to approximately 40 mg As/kg bw (BIBRA 1990). The reported LD_{50}s for the less-soluble arsenic trioxide and calcium arsenate, the virtually insoluble lead arsenate, and the insoluble magnesium arsenate, ranged from 2.2 to 230 mg As/kg bw in rats, mice, rabbits and dogs, while the acute toxicity of insoluble gallium arsenide and nickel subarsenide is much lower (LD_{50}s in the rat of more than 7.8 g As/kg bw and 1 g As/kg bw, respectively) (BIBRA 1990). The lowest reported no-observed-effect level (NOEL) following inhalation in limited available short-term and sub-chronic studies is 1.3 $\mu g/m^3$ arsenic trioxide, based on the observation of slower conditioned reflexes and histological changes in the brain, liver and lungs of rats at the higher concentration (0.005 mg/m^3) (Rozenshtein 1970).

The acute toxicity of ingested inorganic arsenic in humans also increases with the solubility of the compound. The lethal dose in humans is estimated to be approximately 50 to 300 mg (0.8 to 5 mg/kg bw) of arsenic trioxide, although severe effects have been reported following ingestion of as little as 20 mg, and persons have survived oral doses of approximately 10 g (BIBRA 1990).

Chronic and Long Term Toxicity

In studies in experimental animals, the main organs affected by repeated oral doses of arsenic compounds are the liver and kidneys, although effects have also been noted on the spleen, body weight and various haematological and biochemical parameters.

The no-observed adverse-effect level (NOAEL) for chronic ingestion of arsenic appears to be in the region of 1.5 to 3.5 mg As/kg bw/day, based on 2-year studies in which rats and dogs were administered arsenic in the diet (BIBRA 1990). However, toxic effects, such as reduced growth and litter size and histological changes in the liver, kidney, spleen and skin, have occurred at or below 1.5 mg As/kg bw/day when arsenic is administered in the drinking water of rats or mice for up to 2 years (Byron *et al.* 1967; Schroeder and Balassa 1967; Schroeder and Mitchener 1971).

Chronic ingestion of arsenic in humans has been associated with adverse effects on the skin, including hyperkeratosis of the palms and soles, and hyperpigmentation in several epidemiological studies. There has been an exposure-response relationship between concentrations of arsenic in drinking water supplies and the prevalence of non-cancerous dermal lesions in studies in Taiwan and Chile (Tseng *et al.* 1968; Tseng 1977; Borgono and Greiber 1972; Borgono *et al.* 1977; Borgono *et al.* 1980). Similar dermatological effects have been reported in several studies in workers occupationally exposed to airborne arsenic (BIBRA 1990).

Ingestion of arsenic in drinking water has been associated with an increased prevalence of "blackfoot disease," a peripheral vascular disease leading to gangrene of the toes and feet, in a population of 40 421 residents in Taiwan whose well water contained up to 1,820 µg/L arsenic (Tseng *et al.* 1968; Tseng 1977). This effect has not been observed, however, in populations ingesting drinking water containing high concentrations of arsenic in other countries, and it has been suggested that other compounds in the water supply may play a role in the development of the disease (Chen *et al.* 1988; Lu 1990a,b).

Mixed results have been obtained in numerous epidemiological studies designed to investigate the association between exposure to arsenic and mortality due to cardiovascular and cerebrovascular causes (BIBRA 1990; Wu *et al.* 1989), although in most studies in which an increase in mortality due to these causes was reported (Lee and Fraumeni 1969; Wall 1980;Lubin *et al.* 1981; Welch *et al.* 1982), there was no relationship between excess mortality and average concentration of arsenic or duration of exposure.

Carcinogenicity

The results of early, limited carcinogenicity bioassays, in which experimental animals have been administered arsenic compounds by ingestion or inhalation, have been largely negative (U.S. EPA 1984).

In some more recent studies, increases in the incidence of primarily pulmonary adenomas were observed in hamsters following weekly intratracheal instillations of calcium arsenate and arsenic trioxide for 15 weeks (Pershagen and Bjorklund 1985; Yamamoto *et al.* 1987), whereas in other similar studies, results for arsenic trioxide, gallium arsenide and arsenic trisulphide were negative (Ohyama *et al.* 1988; Yamamoto *et al.* 1987; Pershagen and Bjorklund 1985). The results of initiation/promotion studies have provided some limited evidence that some arsenic compounds may act as tumour promoters in experimental animals (Shirachi *et al.* 1986; 1987). Arsenic appears to induce clastogenic damage rather than gene mutations in short-term in vitro and in vivo assays, and induces transformation in mammalian cells in vitro (BIBRA 1990; IARC 1987).

Excesses in mortality due to lung cancer have been associated with exposure to arsenic in numerous analytical epidemiological studies of workers in smelters and arsenical pesticide production facilities, with considerable evidence of an exposure-response relationship. Where it was possible to investigate the influence of possible confounders, such as concomitant exposure to sulphur dioxide or cigarette smoke, these factors have not explained this excess. In addition, in the populations for which exposure has been the most extensively characterized (i.e., three cohorts of workers employed at smelters in Washington, Montana and Sweden), there was a clear exposure-response relationship between airborne arsenic levels and mortality due to lung cancer (Enterline *et al.* 1987; Higgins *et al.* 1986; Jarup *et al.* 1989). Although not as well studied, there is also evidence that cancers at sites other than the lung, including the stomach, colon, liver and urinary system, may be associated with occupational exposure to arsenic (Gibb and Chen 1989).

Associations between the ingestion of arsenic in drinking water, or in the form of a medicinal, and skin cancer have also been observed in numerous case studies, case series and epidemiological investigations in Taiwan (Tseng *et al.* 1968; Tseng 1977; Chen *et al.* 1985), Central and South America (Bergoglio 1964; Biagini *et al.* 1974; Zaldivar 1974; 1977; Cebrian *et al.* 1983) and England (Philipp *et al.* 1983).

The largest and most sensitive epidemiological study was conducted in Taiwan where a population of 40,421 was divided into three groups, based on the arsenic content of their well water (high $\geq$0.60 mg/L, medium 0.30 to 0.59 mg/L and low 0.01 to 0.29 mg/L) (Tseng 1977). There was a clear exposure-response relationship between the arsenic concentration of drinking water and the prevalence of skin cancer (Tseng *et al.* 1968; Tseng 1977). However, HWC (1989) found several methodological weaknesses and potential confounding factors that complicate the interpretation of the results of this investigation. For example, the investigators were not "blinded" as to whether persons being examined were from the arsenic-endemic area. The socioeconomic conditions in the study area are poor, and the population subsists on food somewhat low in protein and fat (Tseng 1977). Exposure to arsenic from sources other than drinking was not also examined, and there is some, albeit not well-documented, indication that these sources may have contributed significantly to the total exposure of the Taiwanese population (Heydorn 1970; Li *et al.* 1979).

There is also more recent evidence of an association between the ingestion of drinking water containing high concentrations of arsenic and increased mortality due to various cancers of internal organs, including the bladder, kidney and lung, in exposed populations in Taiwan (Chen *et al.* 1986; Wu *et al.* 1989). Hepatic angiosarcoma (a rare tumour of the liver) and lung cancer have also been reported in patients ingesting arsenic medicinals (Robson and Jelliffe 1963; Regelson *et al.* 1968; Lander *et al.* 1975; Popper *et al.* 1978; Falk *et al.* 1981; Roat *et al.* 1982).

Immunotoxicity

Possible impairment of the immune system has been reported in an unconfirmed study of a small number of individuals drinking water containing 390 µg/L of arsenic (Ostrosky-Wegman *et al.* 1991). Adverse effects on the immune system, including increased susceptibility to bacterial and viral infection, suppressed humoral response and a reduction in the number of T lymphocytes, have been reported in several studies in which mice were exposed to arsenic by inhalation or ingestion (BIBRA 1990).

Genotoxicity and Related Effects (Extracted from HWC 1992)

Arsenic does not appear to be mutagenic in bacterial and mammalian assays, although it can induce chromosome breakage, chromosomal aberrations and sister chromatid exchange in a linear, dose-dependent fashion in a variety of cultured cell types, including human cells (Jacobson-Kram and Montalbano 1985; U.S. EPA 1988). Most of the chromosomal aberrations are lethal events, so that the cells do not survive more than one or two generations; consequently, the damage caused by arsenic has no genetic consequences. Trivalent arsenic is approximately an order of magnitude more potent than As(V) in this respect. The clastogenic effect of arsenic appears to be due to interference with DNA synthesis, as arsenic induces sister chromatid exchange and chromosomal aberrations only when present during DNA replication. Arsenic has also been shown to block dividing cells in the S and G2 phases (Petres *et al.* 1977).

Neurotoxicity

Although slight neurological effects were reported in a small group of individuals in Nova Scotia whose well water contained more than 0.05 ppm (50 µg/L) arsenic (Hindmarsh *et al.* 1977), no adverse neurological effects were observed in two larger studies of populations exposed in some cases to higher concentrations (Kreiss *et al.* 1983; Southwick *et al.* 1983).

Reproductive Toxicity and Teratogenicity

Little recent information is available on the developmental and reproductive toxicity of arsenic. Foetotoxic effects have been observed at doses that are not maternally toxic (i.e., ≥ 8 mg As/kg bw/day) (Matsumoto *et al.* 1973), but teratogenic effects have been observed only at doses that were toxic to the dams. Available data on the reproductive toxicity of arsenic are limited largely to early studies, in which the lowest concentration reported to induce effects (decreased litter size and increased ratio of male to female offspring in a three generation study in mice) was 1.5 mg As/kg bw/day (Schroeder and Mitchener 1971).

There is no conclusive evidence that arsenic causes any adverse reproductive effects in humans, based on several very limited studies (Nordstrom *et al.* 1978a; 1978b; 1979a; 1979b; Beckman and Nordstrom 1982; Aschengrau *et al.* 1989).

In a case-control study of 270 children with congenital heart disease and 665 healthy children, maternal consumption during pregnancy of drinking water containing detectable arsenic concentrations was associated with a threefold increase in the occurrence of coarction of the aorta (the prevalence odds ratio adjusted for all measured contaminants and source of drinking water was 3.4 with 95% confidence limits of 1.3 to 8.9) (Zierler *et al.* 1988). However, there was no adjustment for maternal age, socioeconomic status or previous reproductive history.

In a case-control study in Massachusetts of 286 women with spontaneous abortions and 1391 women with live births, elevated odds ratios for miscarriages were associated with exposure to arsenic in drinking water (Aschengrau *et al.* 1989). The odds ratios for spontaneous abortion adjusted for maternal age, educational level and history of prior spontaneous abortion for women exposed to arsenic in their drinking water at undetectable concentrations, 0.0008 to 0.0013 mg/L and 0.0014 to 0.0019 mg/L were 1.0, 1.1 and 1.5, respectively. Exposure was determined by matching each woman to the results of a tap water sample taken in her city or town during pregnancy. However, the median interval from the date of matched metal analysis sample to the date of conception was 2.1 years, and it was reported that the variability of concentrations of metals in 20 Massachusetts towns and cities over the seven year period between 1978 and 1985 was 10- to 100-fold.

The following review on the teratogenicity of inorganic arsenic was extracted from HWC 1992. In early studies, teratogenic effects of arsenic in chicks, golden hamsters and mice were reported (Hood and Bishop 1972; Zierler *et al.* 1988). In a more recent investigation (Ferm and Hanlon 1985), arsenate was teratogenic in the offspring of pregnant hamsters following exposure on days 4 to 7 of gestation by mini pump implantation. The threshold blood level for teratogenesis was 4.3 µmol/kg (Hanlon and Ferm 1986a). The specific form of arsenic that is responsible for teratogenesis is not known, although there is evidence to suggest that it is arsenite rather than arsenate (Hanlon and Ferm 1986b). In studies with mice and

hamsters, MMAA (monomethylarsonic acid) and DMAA (dimethylarsinic acid) have been considerably less teratogenic than As(III) or As(V) (Lovell and Farmer 1985).

3.5 Classification and Evaluation

On the basis of documented carcinogenicity in human populations by more than one route of exposure (*i.e.*, both inhalation and ingestion), HWC (1992) and EC/HWC (1993) have classified "the group of inorganic arsenic compounds as a whole" in Group I ("carcinogenic to man") of the classification scheme developed by the Bureau of Chemical Hazards for use in the derivation of the "Guidelines for Canadian Drinking Water Quality" (HWC 1989).

As 77% of Canadians live in cities (Statistics Canada 1993), an urban exposure scenario is the most common situation expected to arise. Given that 82% of these urban dwellers receive treated water supplies, mostly from surface water sources (Tate and Lacelle 1992), then the most likely route of exposure to contaminants in soil is expected to be direct soil ingestion [intentional (i.e. pica) or unintentional], for non-volatile compounds. However, because no risk specific doses have been estimated for direct soil ingestion, risk estimates from the ingestion of drinking water are used, based on an adult daily exposure scenario.

Based on the increased incidence of skin cancer observed in the Taiwanese population (Tseng *et al.* 1968), the U.S. EPA has estimated lifetime skin cancer risks associated with the ingestion of arsenic in drinking water using a multistage model modified to take into account incidence stratified by age group (HWC 1992). On the basis of a review of available data, HWC (1992) has concluded that the EPA model was the most appropriate for estimation of the skin cancer risks associated with the ingestion of arsenic in drinking water. The estimated doses corresponding to lifetime skin cancer risks of 10^{-4}, 10^{-5}, 10^{-6} and 10^{-7} (based on Taiwanese men) were as follows (HWC 1992):

Lifetime Incremental Risk	Dose (µg/kg bw/day)
10^{-4}	0.06
10^{-5}	0.006
10^{-6}	0.0006
10^{-7}	0.00006

However, HWC (1992) emphasised that these estimates probably represent a worst-case situation because of concomitant exposure to other compounds in the water in Taiwan and possible dietary deficiencies of the Taiwanese population compared with North American populations. Moreover, methylation (*i.e.*, detoxification) of inorganic arsenic is progressively but not completely saturated when daily intake of humans exceeds approximately 500 µg, and this has not been taken into consideration in derivation of the quantitative estimates of risk (HWC 1992).

4. DERIVATION OF HUMAN HEALTH SOIL QUALITY GUIDELINES

Human health soil quality guidelines are designed to protect residential/parkland, commercial, and industrial land uses, and the use of ground water as a source of drinking water. The residential/parkland human health soil quality guideline is also applicable to the residential use of agricultural land. It does not, however, account for the potential intake of contaminants via consumption of produce, meat and milk produced and consumed at remediated agricultural sites. The *Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines* (CCME 1996) can be used to establish guidelines for agricultural uses which take into consideration the intake of contaminants from produce, meat, and milk produced and consumed on-site, if desired and where applicable. The following derivations have been based on inorganic forms of arsenic as these forms cause the main toxicity in human receptors.

4.1 Protocol

Inorganic arsenic has been classified in the Group I category of substances (i.e., carcinogenic to man) (HWC 1992; EC/HWC 1993). Thus, it is considered as a non-threshold toxicant (a substance for which there is considered to be some probability of harm for the critical effect at any level of exposure) requiring the development of soil quality guidelines that employ a critical Risk specific Dose (RsD), based on lifetime incremental risks from soil ingestion (CCME 1996). For all land uses, the adult was chosen as the receptor when considering lifetime cancer risk (CCME 1996). The following derivation provides guideline values at estimated lifetime incremental risks from 10^{-4} to 10^{-7} which are applicable to all land uses for which human health soil quality guidelines are derived [*i.e.* , residential/parkland, agricultural (when limited to the residential uses of farm property), commercial and industrial]. The selection of acceptable level of risk is a policy decision. The CCME Subcommittee on Environmental Quality Criteria for Contaminated Sites recommends the development of a soil guideline based on an incremental risk from soil exposure of 10^{-6} (CCME 1996). It should be noted that the 10^{-6} incremental risk above background falls within the range (less than 10^{-5} to 10^{-6}) considered to be "essentially negligible" in the derivation of Maximum Acceptable Concentrations (MACs) for carcinogenic chemicals in drinking water (HWC 1989).

Absorption factors may be required where the critical toxicity study used in developing the cancer slope factor has used an absorbed dose rather than an administered dose or where the critical toxicity study has used a different medium in developing the cancer slope factor than that under investigation. Then soil ingestion rates are multiplied by corresponding Absorption Factors (AFs), when these data are available.

The exposure term for a non-threshold substance is *one* for all land uses since the exposure period (*e.g.*, 10 hrs/day, 5 days/week and 48 weeks/year for 30 to 40 years over a lifetime at commercial and industrial sites) exceeds the likely latency period for most carcinogens.

4.2 **Residential/Parkland, Commercial and Industrial Land Uses** (adult receptor)

Using the above information, a guideline which applies to an adult lifetime cancer risk for urban residential/parkland (and agricultural when limited to residential uses of farm property), commercial and industrial soil exposure can be determined as:

$$IRSQG_{HH} = \frac{RsD \times BW}{AF_i \times SIR \times ET} + BSC$$

where,

$IRSSQG_{HH}$ = Incremental Risk Specific Human Health Soil Quality Guideline for residential /parkland, agricultural (when limited to residential uses of farm property), commerical and industrial settings (µg/g);

RsD = Risk specific Doses (µg/kg bw/day) corresponding to lifetime incremental risks above background (see section 3.5 for doses);

BW = Body Weight for an adult = 70 kg (Health Canada 1994);

AF_i = Absorption Factor from gut (medium specific)[1] = 1 by default;

SIR = Soil Ingestion Rate = 0.02 g/day (Health Canada 1994);

ET = Exposure Term = 1;

BSC = Background Soil Concentration = 10.0 µg/g (see section 2.5 and Table 2b).

Thus, $IRSSQG_{HH}$ =

Lifetime incremental risk above background	$\frac{RsD \times BW}{AF_i \times SIR}$ (µg/g)	Background soil concentration (µg/g)	$IRSSQG_{HH}$ (µg/g)	Total estimated risk from soil at $IRSSQG_{HH}$

[1] This factor accounts for the attenuation of exposure resulting from the absorption of a contaminant to a specific environmental medium (e.g. soil). For practical purposes in this case, this factor is 1 by default, due to lack of data

	10^{-4}	210	10	220	1.1×10^{-4}
	10^{-5}	21	10	31	1.5×10^{-5}
Recommended by CCME (1996)	10^{-6}	2.1	10	12	5.7×10^{-6}
	10^{-7}	0.21	10	10	4.8×10^{-6}

The background concentration for inorganic arsenic, 10 µg/g, was the highest reported mean concentration in urban environments not directly impacted by point sources. The incremental risk specific guidelines for residential/parkland (and agricultural when limited to residential uses of farm property), commercial and industrial land uses may be below soil background levels in some areas of Canada. Where local or regional background soil concentrations of arsenic differ markedly from the 10 µg/g, then site- or region-specific guidelines should be derived, incorporating the local/regional background soil concentrations.

4.3 Use of Ground Water as a Source of Raw Water for Drinking

No guideline for the protection of ground water used as a source of raw water for drinking was derived for inorganic arsenic due to constraints on the mathematical model when applied to inorganic compounds (CCME 1996).

5. CONCLUSION

As a human carcinogen, no specific soil guideline above background is recommended for inorganic arsenic. Human exposure to inorganic arsenic in soil should be reduced to the lowest levels deemed reasonably feasible. Provisional risk specific human health soil quality guidelines of 220, 31, 12 and 10 µg/g (mg/kg) dry soil for all land uses of concern, namely residential/parkland, agricultural (when restricted to the residential uses of a farm property) commercial, and industrial, with associated lifetime risks of 10^{-4}, 10^{-5}, 10^{-6} and 10^{-7} above background, respectively, are proposed.

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Table 1. Identity and Physical and Chemical Properties of Arsenic and Selected Inorganic Arsenic Compounds[1]

Property	Arsenic	Arsenic acid	Arsenic pentoxide	Arsenic trioxide	Calcium arsenate	Gallium arsenide	Sodium arsenate	Sodium arsenite
Empirical formula	As	H_3AsO_4	$As_2O_5(As_4O_{10})$	$As_2O_3(As_4O_6)$	$Ca_3(AsO_4)_2$	GaAs	Na_2HAsO_4	$NaAsO_2$
Molecular wt	74.92	150.95	229.84	197.84	398.08	144.64	185.91	129.91
Valence	0	+5	+5	+3	+5	-3	+5	+3
CAS #	7440-38-2	7778-39-4	1303-28-2	1327-53-3	7778-44-1	1303-00-0	7778-43-0	7784-46-5
Common synonyms	arsenic black, colloidal arsenic, gray arsenic	orthoarsenic acid	arsenic(V) oxide, arsenic acid anhydride, diasernic pentoxide	arsenic oxide, arsenious acid, arsenious oxide, white arsenic	calcium orthoarsenate, arsenic acid, calcium salt	gallium monoarsenide	disodium arsenate, sodium biarsenate, arsenic acid, disodium salt	arsenious acid, sodium salt, sodium metaarsenite
Color, Physical state	gray, solid	white, solid	white, solid	white[d], solid[d]	colorless, solid	dark gray, solid	No data, solid	gray-white, solid
Melting point	817°C @ 28 atm	35.5°C	decomposes @ 315°C	312.3°C	1.455°C	1238°C	57°C	No data
Boiling point	613°C sublimes	loses H_2O @ 160°C	No data	465°C[d]	No data	No data	No data	No data
Density g/cm^3	5.727	2.0-2.5	4.32	3.738	3.62	5.31	1.87	1.87
Log K_{ow}	No data	No data	No data	No data	No data	No data	No data	No data
Log K_{oc}	No data	No data	No data	No data	No data	No data	No data	No data
Vapour pressure	1 mmHg @ 372°C, 40 mmHG @ 483°C, 100 mmHg @ 518°C	No data	No data	66.1 mmHg @ 312°C	No data	No data	No data	No data
Solubility in water	insoluble; soluble in nitric acid	soluble in 3,020 g/L @ 12.5°C	soluble in 1,500 g/L at 16°C, 658 g/L at 20°C, 767 g/L at 100°C; soluble in acid	soluble in 37 g/L at 20°C, 101 g/L at 100°C; soluble in HCl[b]	soluble in 0.13 g/L at 25°c; soluble in dilute acids	No data	soluble	very soluble
Solubility in organic solvents	No data	soluble in alcohol	soluble in alcohol	soluble in glycerin, slightly soluble in alcohol	insoluble	No data	slightly soluble in alcohol	slightly soluble in alcohol

1 Adapted from ATSDR (1992)
2 Value for $H_3AsO_4 \cdot \frac{1}{2}H_2O$

Table 2a. Arsenic Concentrations in Canadian Glacial Till

Sampling Site	Size fraction (mm)	N	Arsenic Content mean ± std dev., (range) µg/g dry wt	Sample preparation/ Analytical method	Reference
Central Newfoundland	<0.002	838	47.5 ± 98.2, (1-2138) median: 24	ICP-AES	Klassen (1994)
Southeastern Cape Breton, N.S.	<0.063	324	(2.5-128) median: <5	ICP-AES	McClenaghan, undated
South Miramichi	<0.002	873	34.2 (0.5-664)	not reported	Lamothe (1990)
	<0.063	873	17 (1-310)		
Central Miramichi	<0.002	1782	38 (1-2000)		
	<0.063	1782	15.2 (0.5-398)		
North Miramichi	<0.002	1368	46 (1-640)		
	<0.063	1368	23.7 (3-201)		
Southeastern Ontario & southwestern Quebec	<0.002	2129	5 ± 18.7, (1-430) median: 2	AAS	Kettles and Shilts (1994)
Northeastern Ontario, east of Timmins and N. of Kirkland Lake	<0.063	279	(0.5-44) median: 1	ICP-AES	McClenaghan (1990)
Manitouwadge-Hornpayne region, Ontario	<0.002	603	9.6 ± 9.9, (1-61) median: 6	ICP-AES hot aqua-regia leach	Kettles (1994)
Beardmore-Geraldton area, east of Lake Nipigon, Ontario	<0.063	505	5.4 ± 7.9, (3-85) median: 3	ICP-AES nitric aqua-regia partial leach	Thorleifson & Kristjansson (1990)
S.W. Manitoba, Bissett-English Brook area & Rice L. Greenstone Belt	<0.002	208	53.7 ± 233.6, (1-2704) median: 14	ICP-AES nitric aqua-regia partial leach	Henderson (1994)
Cormorant Lake Area (Saskatchewan/Manitoba)	<0.002	1591	(1-6590)	ICP-AES nitric aqua-regia partial leach	MacMartin *et al.* (1994)
Manson River/Fort Fraser area, B.C.	<0.002	402	(<5-112); median: 9	ICP-AES nitric aqua-regia partial leach	Plouffe & Ballantyne (1993)
	<0.063	401	(<5-73); median: 5		
	<0.063	400	(1.9-69.8); median: 13	INAA	
Fish Lake area, B.C.	<0.002	78	(0-38); median: 9	ICP-AES nitric aqua-regia partial leach	Plouffe & Ballantyne (1994)
	<0.063	108	(0-7); median: 7		
	<0.063	108	(0.5-31); median: <9	INAA	
Kaminak Lake/Turquetil Lake area, District of Keewatin, NWT	<0.063	2264	(2.4-20); median: <4	ICP-AES nitric aqua-regia partial leach	Shilts and Wyatt (1989)
Aylmer Lake, District of Mackenzie, NWT	<0.002	193	54.5 ± 43.1, (1-224); median: 42	ICP-AES	Dredge *et al.* (1994)
	<0.063	191	11 ± 10.8, (0.25-100) median: 8.2	INAA	
Lac de Gras, District of Mackenzie, NWT	<0.002	183	59.4 ± 34.2, (1-154); median: 54	ICP-AES	Ward *et al.* (1994)
	<0.063	178	3.8 ± 0.29, (1.4-25); median: 7.7	INAA	

Table 2a. Arsenic Concentrations in Canadian Glacial Till (Cont'd)

Sampling Site		Size fraction (mm)	N	Arsenic Content mean ± std dev., (range) µg/g dry wt	Sample preparation/ Analytical method	Reference
Winter Lake, District of Mackenzie, NWT		<0.002	120	36.4 ± 31.1, (4-200); median: 28	ICP-AES	Kerr *et al.* (1994)
		<0.063	115	4.4 ± 3.7, (0.8-29); median: 3.4	INAA	
S. Melville Peninsula, NWT	1990	<0.002	280	104 ± 14.7, (2.5-207); median: 8	ICP-AES	Dredge (1995)
	1991	<0.002	255	15.5±43.8, (1-520); median: 2	ICP-AES	

KEY: N = number of samples; ICP= Inductively Coupled Plasma; MS= Mass Spectrometry; AAS= Atomic Absorption Spectrometry; AES= Atomic Emission Spectrometry; INAA = Instrumental Neutron Activation Analysis.

Table 2b. Background Concentrations of Arsenic in Canadian Surface Soils

Sampling Site	Number of samples	Arsenic content: mean, (range) µg/g dry wt	Sample preparation/ Analytical method	Reference
Ontario - Central & Southern (soil geochemical survey (<2 mm fraction, milled to 100 µm)	300	3.72±3.57 (<0.5-30) (median: 2.8)	$HF/HClO_4/HNO_3$ INAA	Sharpe and Rasmussen (1996)
Ontario - uncontaminated agricultural soils	207	6.3 (1.1-16.7)	Chromogenic complex (arsine generation) spectrophotometry	Frank *et al.* (1976)
Ontario - old urban parklands	60	5.9 (<0.2-60.7)	hydride generation / Flameless AAS	OMEE (1994a)
Ontario - rural parklands	101	3.9 (<0.2-30)	hydride generation / Flameless AAS	OMEE (1994a)
Ontario - Halton region agricultural soils (no sludge treatment)	252	range of means: 5.1-6.0	hydride generation / flameless AAS	Webber and Shamess (1987)
Ontario - agricultural soils (no sludge treatment) from 10 locations	[a]	overall mean: 4.2 (range of means: 1.1-7.2)	calorimetry	Webber *et al.* (1981)
Ontario - Toronto, urban background	not reported	9.8 (2.7 - 41)	hydride method/AAS	Temple *et al.* (1977)
Ontario - southern, agricultural soils	26 sites	5.1, (1.7 - 8.5)	arsine generation from HNO_3-H_2SO_4/flameless AAS	Whitby *et al.* (1978)
Ontario - rural and agricultural soils	not reported	(range of means: 1.3-13.5)	not reported	OMAF (1989)
Ontario - Windsor area, 1990 - urban - rural	 12 sites[b] 18 sites[b]	 7.2 (4.1 - 16.7) 5.8 (4.8 - 7.2)	not reported	Gizyn (1994)
Canada - literature review	not reported	range of means: (4.8-13.6)	not reported	Kabata-Pendias & Pendias (1992)
Québec - Noranda background level in peat	not reported	1.9	not reported	Azzaria and Frechette (1987)
B.C. - Nelson - reference site	≈ 100	5	$HNO_3/HF/HClO_4$ digestion, ICP/AES	Lynch *et al.* (1980)
B.C. - Vancouver background level	not reported	3	not reported	Henning and Konasewich (1984)
Manitoba, Saskatchewan and Alberta surface soils	1273	6.6 (0.25-19) median: 6.6	INAA	R. Garrett, NRCan, pers. com.
Alberta - background concentration	78	(0.40-7.1), most samples in the 4.0-6.5 range	graphite furnace AAS	Dudas and Pawluk (1980)
New Brunswick - 1983-84 survey	40 sites	8.9 (2.3-34.9)	not reported	MacMillan (1985)
New Brunswick - 1992, residential garden soils - East St John[c] - West St John[c] - Fredericton	 18 4 2	 9.0 ± 1.5 12.7± 2.4 10.7± 0.4	not reported	Pilgrim (1993)

KEY: N = number of samples; ICP= Inductively Coupled Plasma; AAS= Atomic Absorption Spectrometry; AES= Atomic Emission Spectrometry; INAA = Instrumental Neutron Activation Analysis.

a 20 cores per composite sample from 4 areas per location
b 3 replicates per site
c In addition to naturally high background levels of As in soils, several potential point sources, such as the refinery and NB power generators, may be sources of As in soils in St John. The weighted mean concentration for the 2 data sets is 9.7 µg As/g dry soil

Table 2c. Arsenic Concentrations in Contaminated Canadian Soils

Sampling Site	Number of Samples	Arsenic Content mean, (range) µg/g dry wt	Sample preparation/ Analytical method	Reference
NWT - Yellowknife town site - near mines	not reported	(1-524) max 7600	XFS	CPHA (1977)
B.C. - vicinity of Pb/Zn smelter in Trail	≈ 100	49	$HNO_3/HF/HClO_4$ digestion, ICP-AES	Lynch *et al.* (1980)
B.C. - vicinity of Trail Pb/Zn smelter in 1982 - 41 park samples in 1989	4 sites 41	20, (8-43) 18, (<1-50)	not reported	H. Teindl, pers. com.
Trail, B.C. - residential soils in the vicinity of Pb/Zn smelter in 1989	122	48, (33-352)	ultrasonic nebulization - ICP-AES	Hertzman *et al.* (1991)
Manitoba - near Flin Flon smelter	not reported	(65-280)	cold-vapour/AES	Zoltai (1988)
Ontario - within 26 km radius from a nickel/copper smelter in Sudbury	not reported	10, (3-47)	not reported	McGovern and Balsillie (1975)
Québec - peat samples within 15 km of Noranda smelter	not reported	9	not reported	Azzaria and Frechette (1987)
Québec - humus samples near site of old gold roasting plant in Duparquet - peat samples within a few km of roasting site	not reported	110 max 1000	not reported	Azzaria and Frechette (1987)
New Brunswick - near smelter	not reported	(5-66.3)	not reported	M. Murphy, pers. com.
New Brunswick - garden soil within 1.9-15.07 km of smelter	not reported	(7.8-38.5)	not reported	Brunswick Mining and Smelting Corporation
B.C.: Burnaby, near municipal incinerator (1987-90) - residential gardens (2 sites) - dairy pasture - blueberry patch - produce farm using commercial fertilizers - cranberry patch - bog - woodland park	 48 24 24 24 24 18 18	 5.5, (2.8-8.8) 8.7, (6.2-15.2) 4.1, (1.0-14.5) 6.1, (4.3-10.6) 2.2, (0.4-4.4) 5.4, (2.3-3.9) 8.2, (4.5-6.8)	hydride generation/IPC-AES	Soilcon (1990)
Ontario - Toronto - near a secondary smelter - near power station with previous coal burning activity - near a municipal incinerator - near a major expressway - near a sewage sludge incinerator	not reported	 70, max 2000 25, (6.8 - 114) 16, (3.0 - 49) (3.0 - 24) (4.2 - 20)	hydride method/AAS	Temple *et al.* (1977)
Ontario - Halton Region sludge treated agricultural soils	57	5.7 (3.7-8.1)	nitric/sulfuric acid dig. + hydride generation Flame/flameless AAS	Webber and Shamess (1987)
Ontario - sludge treated agricultural soils from 10 locations	*	4.6 (1.5-6.3)	calorimetry	Webber *et al.* (1981)
Ontario - agricultural soils where arsenical pesticides were used	89	26.5 (1.7-91.6)	Chromogenic complex spectrophotometry	Frank *et al.* (1976)
Southwestern Ontario - old apple orchards sprayed with arsenical pesticides	not reported	(45-290)	not reported	Boyle and Jonasson (1973)
Nova Scotia - old apple orchards sprayed with arsenical pesticides	not reported	(10-124)	not reported	Boyle and Jonasson (1973)
New Brunswick - wood preservation facilities	not reported	max: 10860	not reported	Bamwoya *et al.* (1991)

KEY: N = number of samples; ICP= Inductively Coupled Plasma; MS= Mass Spectrometry; AAS= Atomic Absorption Spectrometry; AES= Atomic Emission Spectrometry; XFS= X-ray Fluorescence Spectrometry

* 20 cores per composite sample from 4 areas per location

Table 3. Existing Criteria and Guidelines for Arsenic in Soil and Ground Water from Various Jurisdictions

Jurisdiction	Description	Maximum concentration		Reference
		Soil (µg/g dry wt)	Ground water (µg/L)	
Canada	Interim Assessment Criterion	5	5	CCME (1991)
	Interim Remediation Criteria	Agr : 20 R/P : 30 C/I : 50		
	Interim maximum acceptable concentration for drinking water		25	HWC (1992)
British Columbia	Clean-Up Criteria	A : 5 B : 30 C : 50	A : 5 B_{DW} : 50 B_{DS} : 50	BC MOE (1989)
Alberta	Tier 1 Soil Criterion	10	-	Alberta Environment (1990)
Ontario	Proposed Clean-up Criteria	Potable & non-potable ground water situations: Agr : 20 R/P : 20 C/I : 20	all land use categories: potable gw: 25 non-potable gw: 480	OMEE (1994b)
Québec	Guidelines	A : 10 B : 20 C : 30	A : 5 B : 50 C : 100	MENVIQ (1988)
Massachusetts	Proposed Soil standards S-1/GW-1, S-1/GW-2, S-1/GW-3[a] S-2/GW-1, S-2/GW-2, S-2/GW-3[b] S-3/GW-1, S-3/GW-2, S-3/GW-3[c]	 30 30 30	Proposed Groundwater Standards GW-1: 50 GW-2: – GW-3: 400	MDEP (1994)
New Jersey	Proposed Surface Soil Standards (based on direct soil contact)	residential: 2 non-residential: 2 impact to ground water: -	Class IIA: 8	NJDEP (1992)
The Netherlands	Target value Intervention value	29 55	10 60	MHSPE (1994)
U.K.	Threshold Trigger Values	domestic gardens: 10 parklands : 40	-	U.K. DOE (1990)
Australia	Interim Acceptable Criteria	20	-	ANZEC (1990)
World Health Organization	Provisional drinking water guideline	-	10	WHO (1993)
Health Canada	Interim Maximum Acceptable Concentration (IMAC)	-	25	HWC (1992)

Key: Agr: agricultural land use; R/P: residential and parkland land use; C/I: commercial and industrial land use;
B_{DW}: remediation criterion for ground water intended for human consumption; B_{DS}: remediation criterion for water-based discharges

a Concentration based upon sensitive uses of the property and accessible soil (0-3 feet, unpaved), either currently or in the foreseeable future. Additional criteria are established for the protection of ground water (GW-1, ground water used as drinking water; GW-2, potential for volatile materials to migrate into indoor air; GW-3, environmental considerations for protection of surface water), based upon leaching from the soils.

b Concentrations based upon property uses associated with moderate exposure (children present) and accessible soils, either currently or in the foreseeable future. Additional criteria are established for the protection of ground water (GW-1, ground water used as drinking water; GW-2, potential for volatile materials to migrate into indoor air; GW-3, environmental considerations for protection of surface water), based upon leaching from the soils.

c Concentrations based upon restricted access to property with limited potential for exposure, either currently or in the foreseeable future. Additional criteria are established for the protection of ground water (GW-1, ground water used as drinking water; GW-2, potential for volatile materials to migrate into indoor air; GW-3, environmental considerations for protection of surface water), based upon leaching from the soils.

Table 4. Estimated Average Daily Intake of Inorganic Arsenic by the Canadian General Population (Adapted from Hughes *et al.* 1994)

Medium	Estimated Daily Intake (µg/kg bw/day)				
	Infants 0 - 6 mo.[1]	Preschoolers 7 mo.- 4 yr[2]	School age children 5 - 11 yr[3]	Teenagers 12 - 19 yr[4]	Adults 20 - 70 yr[5]
Water[6]	0.5	0.3	0.2	0.1	0.1
Food[7]	0.2	0.3	0.2	0.1	0.08
Air[8]	0.0003	0.0004	0.0004	0.0004	0.0003
Soil/Dirt[9]	0.029	0.062	0.007	0.004	0.003
Total	**0.729**	**0.662**	**0.407**	**0.204**	**0.183**
Cigarette Smoking[10]				0.01 - 0.04	0.01 - 0.03

1 Infants are assumed to weigh 7 kg, breathe 2 m^3 of air per day, drink 0.75 L of water per day (Health Canada 1994) and ingest 20 mg of soil per day (Angus Environmental Limited 1991).

2 Preschoolers are assumed to weigh 13 kg, breathe 5 m^3 of air per day, drink 0.8 L of water per day (Health Canada 1994) and ingest 80 mg of soil per day (Angus Environmental Limited 1991).

3 School age children are assumed to weigh 27 kg, breathe 12 m^3 of air per day, drink 0.9 L of water per day (Health Canada 1994) and ingest 20 mg of soil per day (Angus Environmental Limited 1991).

4 Teenagers are assumed to weigh 57 kg, breathe 21 m^3 of air per day, drink 1.3 L of water per day and ingest 20 mg of soil per day (Health Canada 1994)

5 Adults are assumed to weigh 70 kg, breathe 23 m^3 of air per day, drink 1.5 L of water per day, and ingest 20 mg of soil per day (Health Canada 1994).

6 Estimates are based on a mean concentration of 5 µg/L measured as total arsenic; levels in most Canadian surface drinking-water supplies are considerably less than this value, although concentrations in ground water often exceed 5 µg/L (Environment Canada 1989a; 1989b; 1989c; 1989d; OME 1989; Manitoba Environment 1989).

7 Estimates are based on mean concentrations of total arsenic detected in various food composites in a Total Diet Study conducted in 6 Canadian cities (mean concentrations ranged from 0.4 ng/g in applesauce to 3048 ng/g in marine fish) (Dabeka *et al.* 1993), food consumption patterns data for various age groups in Canada (Health Canada 1994) and limited information on the percentage of the total arsenic in various food groups which is inorganic (37%) (EC/HWC 1993). Insufficient data were identified to estimate intake of arsenic by infants in breast milk.

8 Estimates are based on the mean airborne arsenic concentration of 0.001 µg/m^3 associated with PM_{10} in most Canadian cities surveyed by Environment Canada (Dann 1994). Since data on levels of arsenic in indoor air are limited, it is assumed that levels of arsenic in ambient air are comparable to those measured in ambient sir.

9 Estimates are based on the upper value of mean arsenic levels, 10.0 µg/g, determined at various uncontaminated urban sites (see Table 2b). It is assumed that all of the arsenic present in soils is inorganic. Since data on levels of arsenic in housedust are limited, it is assumed that levels of arsenic in dust are comparable to those measured in soils.

10 Estimates are based on estimated arsenic content of mainstream cigarette smoke ranging from 40 to 120 ng per cigarette (U.S. DHHS 1989) and 20 cigarettes smoked per day.